Refine Search

Your wildcard search against 10000 terms has yielded the results below.

Your result set for the last L# is incomplete.

The probable cause is use of unlimited truncation. Revise your search strategy to use limited truncation.

Search Results -

Terms	Documents
L3 and treat$	85

Database:
US Pre-Grant Publication Full-Text Database
US Patents Full-Text Database
US OCR Full-Text Database
EPO Abstracts Database
JPO Abstracts Database
Derwent World Patents Index
IBM Technical Disclosure Bulletins

Search: L4

Refine Search | Recall Text | Clear | Interrupt

Search History

DATE: Tuesday, April 11, 2006 Printable Copy Create Case

Set Name side by side	Query	Hit Count	Set Name result set
DB=PGPB,USPT,USOC,EPAB,JPAB,DWPI,TDBD; PLUR=NO; OP=OR			
L4	L3 and treat$	85	L4
L3	ku70 and (antisense or ribozyme)	98	L3
L2	L1 and treat$	10	L2
L1	ku70 same (antisense or ribozyme)	14	L1

END OF SEARCH HISTORY

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     S3      10  S2 AND TREAT?
? show files;ds;t/3,k/all
File   5:Biosis Previews(R) 1969-2006/Apr W1
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         (c) 1999 AAAS
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         (c) 2006 CAB International
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         (c) 2006 Contains copyrighted material
File 156:ToxFile 1965-2006/Mar W3
         (c) format only 2006 Dialog
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File 162:Global Health 1983-2006/Mar
         (c) 2006 CAB International
File 305:Analytical Abstracts 1980-2006/Apr W2
         (c) 2006 Royal Soc Chemistry
File 393:Beilstein Abstracts 2005/Q3
         (c) Beilstein GmbH
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         (c) 2006 ProQuest Info&Learning
File  91:MANTIS(TM) 1880-2006/Feb
         2006 (c) Action Potential
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         (c) 2006 The Gale Group
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         (c) format only 2002 Dialog
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          (c) 2006 BLHCIS
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         (c) 2006 Mass. Med. Soc.
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         (c) 2001 Informania Ltd.

Set     Items   Description
S1         70   KU70 (S) (ANTISENSE OR RIBOZYME?)
S2         24   RD  (unique items)
S3         10   S2 AND TREAT?
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? s ku70 (s) (antisense or ribozyme?)
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         215746  ANTISENSE
          44352  RIBOZYME?
      S1    70  KU70 (S) (ANTISENSE OR RIBOZYME?)
? rd

>>>Duplicate detection is not supported for File 393.

>>>Records from unsupported files will be retained in the RD set.
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? s s2 and treat?
Processing
Processed  10 of  40 files ...
Processing
Completed processing all files
             24  S2
       17111212  TREAT?
      S3    10  S2 AND TREAT?
? show files;ds;t/3,k/all
File   5:Biosis Previews(R) 1969-2006/Apr W1
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File 434:SciSearch(R) Cited Ref Sci 1974-1989/Dec
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File 91:MANTIS(TM) 1880-2006/Feb
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File 149:TGG Health&Wellness DB(SM) 1976-2006/Mar W4
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File 159:Cancerlit 1975-2002/Oct
(c) format only 2002 Dialog
File 164:Allied & Complementary Medicine 1984-2006/Apr
(c) 2006 BLHCIS
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Set	Items	Description
S1	70	KU70 (S) (ANTISENSE OR RIBOZYME?)
S2	24	RD (unique items)
S3	10	S2 AND TREAT?

>>>KWIC option is not available in file(s): 399

3/3,K/1 (Item 1 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0015354690 BIOSIS NO.: 200510049190
The life and death of DNA-PK
AUTHOR: Collis Spencer J (Reprint); DeWeese Theodore L; Jeggo Penelope A; Parker Antony R
AUTHOR ADDRESS: Canc Res UK, Clare Hall Labs, DNA Damage Response Lab, S Mimms EN6 3LD, Herts, UK**UK
AUTHOR E-MAIL ADDRESS: spencer.collis@cancer.org.uk
JOURNAL: Oncogene 24 (6): p949-961 FEB 3 05 2005
ISSN: 0950-9232
DOCUMENT TYPE: Article; Literature Review
RECORD TYPE: Abstract
LANGUAGE: English

...ABSTRACT: microhomology) or DNA ends of no homology. The core components of mammalian NHEJ are the catalytic subunit of DNA protein kinase (DNA-PKcs), Ku subunits *Ku70* and Ku80, Artemis, XRCC4 and DNA ligase IV. DNA-PK is a nuclear serine/threonine protein kinase that comprises a catalytic subunit (DNA-PKcs), with...

...the high levels of unrepaired DSBs after genotoxic insult. Recently, DNA-PK has emerged as a suitable genetic target for molecular therapeutics such as siRNA, *antisense* and novel inhibitory small molecules. This review encompasses the recent literature regarding the role of DNA-PK in the protection of genomic stability and focuses...

...and directed molecular targeting techniques. This review promotes the inhibition of DNA-PK as a valid approach to enhance the tumor-cell-killing effects of *treatments* such as IR.

3/3,K/2 (Item 2 from file: 5)
DIALOG(R)File 5:Biosis Previews(R)

0014365539 BIOSIS NO.: 200300323835
Adenovirus-mediated heat-activated *antisense* *Ku70* expression radiosensitizes tumor cells in vitro and in vivo.
AUTHOR: Li Gloria C (Reprint); He Fuqui; Shao Xiyun; Urano Muneysau; Shen Lingbo; Kim Dooha; Borrelli Michael; Leibel Steven A; Gutin Philip H; Ling C Clifton
AUTHOR ADDRESS: Memorial Sloan Kettering Cancer Center, 1275 York Avenue, New York, NY, 10021, USA**USA
JOURNAL: Cancer Research 63 (12): p3268-3274 June 15, 2003 2003
MEDIUM: print
ISSN: 0008-5472 _(ISSN print)
DOCUMENT TYPE: Article
RECORD TYPE: Abstract
LANGUAGE: English

Adenovirus-mediated heat-activated *antisense* *Ku70* expression **radiosensitizes tumor cells in vitro and in vivo.**

ABSTRACT: *Ku70* is one component of a protein complex, *Ku70* and Ku80, that functions as a heterodimer to bind DNA double-strand breaks and activates DNA-dependent protein kinase. Our previous study with *Ku70*-/- and Ku80-/- mice, and cell lines has shown that *Ku70*- and Ku80-deficiency compromises the ability of cells to repair DNA double-strand breaks, increases radiosensitivity of cells, and enhances radiation-induced apoptosis. In this study, we examined the feasibility of using adenovirus-mediated, heat-activated expression of *antisense* *Ku70* RNA as a gene therapy paradigm to sensitize cells and tumors to ionizing radiation. First, we performed experiments to test the heat inducibility of heat...

...fluorescent protein (EGFP) under the control of an inducible hsp70 promoter, into exponentially growing cells. At 24 h after infection, cells were exposed to heat *treatment*, and heat-induced EGFP expression at different times was determined by flow cytometry. Our data clearly show that heat shock at 42degreeC, 43degreeC, or 44degreeC...

...to be equally effective in activating the hsp70 promoter-driven EGFP expression (>300-fold) in various tumor cells. Second, we have generated adenovirus vectors containing *antisense* *Ku70* under the control of an

inducible hsp70 promoter. Exponentially growing cells were infected with the adenovirus vector, heat shocked 24 h later, and the radiosensitivity determined 12 h after heat shock. Our data show that heat shock induces *antisense* *Ku70* RNA, reduces the endogenous *Ku70* level, and significantly increases the radiosensitivity of the cells. Third, we have performed studies to test whether *Ku70* protein level can be down-regulated in a solid mouse tumor (FSa-II), and whether this results in enhanced radiosensitivity in vivo, as assessed by in vivo/in vitro colony formation and by the tumor growth delay. Our data demonstrate that heat-shock-induced expression of *antisense* *Ku70* RNA attenuates *Ku70* protein expression in FSa-H tumors, and significantly sensitizes the FSa-II tumors to ionizing radiation. Taken together, our results suggest that adenovirus-mediated, heat-activated *antisense* *Ku70* expression may provide a novel approach to radiosensitize human tumors.
DESCRIPTORS:
CHEMICALS & BIOCHEMICALS: heat-activated *antisense* *Ku70*--

3/3,K/3 (Item 1 from file: 34)
DIALOG(R)File 34:SciSearch(R) Cited Ref Sci

13800819 Genuine Article#: 912ZB No. References: 147
Title: Induction of apoptosis by curcumin and its implications for cancer therapy
Author(s): Karunagaran D (REPRINT) ; Rashmi R; Kumar TRS
Corporate Source: Rajiv Gandhi Ctr Biotechnol,Canc Biol Lab,Thiruvananthapuram 695014/Kerala/India/ (REPRINT); Rajiv Gandhi Ctr Biotechnol,Canc Biol Lab,Thiruvananthapuram 695014/Kerala/India/(dkarunagaran@hotmail.com)
Journal: CURRENT CANCER DRUG TARGETS, 2005, V5, N2 (MAR), P117-129
ISSN: 1568-0096 Publication date: 20050300
Publisher: BENTHAM SCIENCE PUBL LTD, EXECUTIVE STE Y26, PO BOX 7917, SAIF ZONE, 1200 BR SHARJAH, U ARAB EMIRATES
Language: English Document Type: REVIEW (ABSTRACT AVAILABLE)

...Abstract: including ourselves, have demonstrated the involvement of several pro and antiapoptotic molecules in curcumin-induced apoptosis, and ways to sensitize chemoresistant cancer cells to curcumin *treatment*. This review describes the mechanisms of curcumin-induced apoptosis currently known, and suggests several potential strategies that include down-regulation of antiapoptotic proteins by antisense...

3/3,K/4 (Item 1 from file: 135)
DIALOG(R)File 135:NewsRx Weekly Reports

0000187953 (USE FORMAT 7 OR 9 FOR FULLTEXT)
Researchers provide details of new studies and findings in the area of oncology
Cancer Weekly, January 25, 2005, p.214

DOCUMENT TYPE: Expanded Reporting LANGUAGE: English
RECORD TYPE: FULLTEXT
WORD COUNT: 1151

... their role in curcumin-induced apoptosis, human colon cancer cells (SW480) were made to over-express or under-express Bcl-XL (by stable transfection) and *Ku70* (by transient transfection) using plasmid constructs that express their genes in sense or *antisense* orientation,

respectively," reported R. Rashmi and colleagues, Rajiv Gandhi Center for Biotechnology, Division of Cancer Biology.

"Stable cells that express Bax [Bax-GFP (green fluorescent...

...in both AsBcl-XL and AsKu70 cells, suggesting a possible feedback activation of caspase 8 by caspase 3.

"Bax-GFP cells were highly sensitized when *Ku70* was down-regulated, supporting the reported role of *Ku70* in the retention of Bax within the cytosol. The study reveals the potential of *antisense* inhibition of antiapoptotic proteins as an effective strategy to tackle chemoresistant cancers with curcumin," researchers concluded.

Rashmi and colleagues published their study in Carcinogenesis (Ectopic ...

...as a target for therapy in order to prevent metastasis," Espana and coauthors suggested.

Espana and colleagues published their study in Breast Cancer Research and *Treatment* (Overexpression of Bcl-xL in human breast cancer cells enhances organ-selective lymph node metastasis. Breast Cancer Res *Treat*, 2004;87(1):33-44).

For more information, contact A. Sierra, Ciutat Sanitaria & University Bellvitge, Hospital Duran & Reynals, Institute Recerca Oncology, Center Molecular Oncology, Gran...

3/3,K/5 (Item 1 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0364800 DBR Accession No.: 2005-10504
The life and death of DNA-PK - DNA-protein-kinase, antisense DNA, RNA interference for use in cancer gene therapy; a review
AUTHOR: COLLIS SJ; DEWEESE TL; JEGGO PA; PARKER AR
CORPORATE AFFILIATE: Johns Hopkins Univ Johns Hopkins Univ Johns Hopkins Univ Univ Sussex
CORPORATE SOURCE: Collis SJ, Canc Res UK, Clare Hall Labs, DNA Damage Response Lab, S Mimms EN6 3LD, Herts, England
JOURNAL: ONCOGENE (24, 6, 949-961) 2005
ISSN: 0950-9232
LANGUAGE: English

...ABSTRACT: microhomology) or DNA ends of no homology. The core components of mammalian NHEJ are the catalytic subunit of DNA protein kinase (DNA-PKcs), Ku subunits *Ku70* and Ku80, Artemis, XRCC4 and DNA ligase IV. DNA-PK is a nuclear serine/threonine protein kinase that comprises a catalytic subunit (DNA-PKcs), with...

... the high levels of unrepaired DSBs after genotoxic insult. Recently, DNA-PK has emerged as a suitable genetic target for molecular therapeutics such as siRNA, *antisense* and novel inhibitory small molecules. This review encompasses the recent literature regarding the role of DNA-PK in the protection of genomic stability and focuses...

... and directed molecular targeting techniques. This review promotes the inhibition of DNA-PK as a valid approach to enhance the tumor-cell-killing effects of *treatments* such as IR. (13 pages)

3/3,K/6 (Item 2 from file: 357)
DIALOG(R)File 357:Derwent Biotech Res.

0350419 DBR Accession No.: 2004-22711 PATENT

Novel non-human mammal having prostate cell containing genome comprising prostate cancer-related oncogene and cancer-prone genetic predisposition, useful for identifying therapeutic agent to prevent or *treat* prostate cancer - a disease model for prostate cancer pharmacogenetics comprises a transgenic mouse having a genetic predisposition to development of cancer

AUTHOR: HEYER J; CHIN L; DEPINHO R A; ALSON S

PATENT ASSIGNEE: GENPATH PHARM INC 2004

PATENT NUMBER: WO 200477942 PATENT DATE: 20040916 WPI ACCESSION NO.: 2004-662331 (200464)

PRIORITY APPLIC. NO.: US 451331 APPLIC. DATE: 20030228

NATIONAL APPLIC. NO.: WO 2004US6118 APPLIC. DATE: 20040227

LANGUAGE: English

...**human mammal having prostate cell containing genome comprising prostate cancer-related oncogene and cancer-prone genetic predisposition, useful for identifying therapeutic** agent to prevent or *treat* prostate cancer **- a disease model for prostate cancer pharmacogenetics comprises a transgenic mouse having a genetic predisposition to development of cancer**

...ABSTRACT: both the oncogene and cancer-prone genetic mutation is inducible. The disabling mutation is accomplished by post-transcriptional silencing, which is accomplished by RNA interference, *antisense* or *ribozyme*. The oncogene is chosen Akt1, Akt2, Akt3, an activating variant of Akt1, Akt2, Akt3, a gene in the PI3K-AKT signal transduction pathways, ras, jun...

...and NKX3.1. The proto-oncogene is chosen from CTNNB1, myc, ras and her2. The DNA repair gene is chosen from MSH2, MSH3, MSH6, PMS2, *Ku70*, Ku80, DNA/PK, ATR, ATM, XRCC4 and MLH1. The prostate cancer-related gene is chosen from p27Kip1, MXI1 and androgen receptor gene. The mammal is...

...USE - (I) which is a mouse chosen A/J, C3H, C57BL/6, FVB, 129 and Balb/C is useful for identifying a therapeutic agent to *treat* prostate cancer, by administering a candidate compound to (I) that has developed prostate cancer, and observing the effect of the compound on cancer development, or...

3/3,K/7 (Item 3 from file: 357)

DIALOG(R)File 357:Derwent Biotech Res.

0316002 DBR Accession No.: 2003-17142

Adenovirus-mediated heat-activated *antisense* *Ku70* expression radiosensitizes tumor cells in vitro and in vivo - recombinant adeno **virus vector plasmid pJM-17-mediated gene transfer and expression in fibroblast cell or HEK-293 for use in cancer gene therapy**

AUTHOR: LI GC; HE FQ; SHAO XY; URANO M; SHEN LB; KIM D; BORRELLI M; LEIBEL SA; GUTIN PH; LING CC

CORPORATE AFFILIATE: Mem Sloan Kettering Canc Ctr Mem Sloan Kettering Canc Ctr Mem Sloan Kettering Canc Ctr; William Beaumont Hosp

CORPORATE SOURCE: Li GC, Mem Sloan Kettering Canc Ctr, Dept Med Phys, 1275 York Ave, New York, NY 10021 USA

JOURNAL: CANCER RESEARCH (63, 12, 3268-3274) 2003

ISSN: 0008-5472

LANGUAGE: English

Adenovirus-mediated heat-activated *antisense* *Ku70* expression radiosensitizes tumor cells in vitro and in vivo - recombinant adeno virus vector plasmid pJM-17-mediated gene transfer and expression in fibroblast cell or...

ABSTRACT: AUTHOR ABSTRACT - *Ku70* is one component of a protein complex, *Ku70* and Ku80, that functions as a heterodimer to bind DNA double-strand breaks and activates DNA-dependent protein kinase. Our previous study with *Ku70* -/- and Ku80-/- mice, and cell lines has shown that *Ku70*- and Ku80-deficiency compromises the ability of cells to repair DNA double-strand breaks, increases radiosensitivity of cells, and enhances radiation-induced apoptosis. In this study, we examined the feasibility of using adenovirus-mediated, heat-activated expression of *antisense* *Ku70* RNA as a gene therapy paradigm to sensitize cells and tumors to ionizing radiation.First, we performed experiments to test the heat inducibility of heat...

... fluorescent protein (EGFP) under the control of an inducible hsp70 promoter, into exponentially growing cells. At 24 h after infection, cells were exposed to heat *treatment* , and heat-induced EGFP expression at different times was determined by flow cytometry. Our data clearly show that heat shock at 42degreesC, 43degreesC, or 44degreesC...

... be equally effective in activating the hsp70 promoter-driven EGFP expression (andgt;300-fold) in various tumor cells. Second, we have generated adenovirus vectors containing *antisense* *Ku70* under the control of an inducible hsp70 promoter. Exponentially growing cells were infected with the adenovirus vector, heat shocked 24 h later, and the radiosensitivity determined 12 h after heat shock. Our data show that heat shock induces *antisense* *Ku70* RNA, reduces the endogenous *Ku70* level, and significantly increases the radiosensitivity of the cells. Third, we have performed studies to test whether *Ku70* protein level can be down-regulated in a solid mouse tumor (FSa-II), and whether this results in enhanced radiosensitivity in vivo, as assessed by in vivo/in vitro colony formation and by the tumor growth delay. Our data demonstrate that heat-shock-induced expression of *antisense* *Ku70* RNA attenuates *Ku70* protein expression in FSa-II tumors, and significantly sensitizes the FSa-II tumors to ionizing radiation.Taken together, our results suggest that adenovirus-mediated, heat-activated *antisense* *Ku70* expression may provide a novel approach to radiosensitize human tumors. (7 pages)

3/3,K/8 (Item 4 from file: 357)

DIALOG(R)File 357:Derwent Biotech Res.

0314222 DBR Accession No.: 2003-15362 PATENT

Predicting whether cancer cells would respond to therapies mediated through Bax-regulated apoptosis, comprises examining the intensity of Bax mRNA or protein expression in cancer cells relative to a control - vector-mediated antisense RNA transfer and expression profiling useful for cancer gene therapy and diagnosis

AUTHOR: MATSUYAMA S; SUN W
PATENT ASSIGNEE: BLOOD CENT RES FOUND 2003
PATENT NUMBER: WO 200327237 PATENT DATE: 20030403 WPI ACCESSION NO.: 2003-371914 (200335)
PRIORITY APPLIC. NO.: US 378585 APPLIC. DATE: 20020508

NATIONAL APPLIC. NO.: WO 2002US29737 APPLIC. DATE: 20020919
LANGUAGE: English

...ABSTRACT: predicting whether the cells will respond to therapies which are mediated through Bax-regulated apoptosis, where a high Bax level indicates that one may lower *Ku70* levels and increase sensitivity to apoptosis, is new. DETAILED DESCRIPTION - INDEPENDENT CLAIMS are included for the following: (1) sensitizing cells to cancer therapy, comprising reducing the cells' native *Ku70* protein level sufficiently so that the cell is more sensitive to cancer therapy; and (2) *treating* cell death-related diseases, comprising increasing cellular *Ku70* protein level in cells sufficiently so that the cells are more resistant to cytotoxic stimuli. BIOTECHNOLOGY - Preferred Method: In predicting whether cancer cells would respond to therapies that are mediated through Bax-regulated apoptosis, one additionally examines the intensity of expression of the *Ku70* gene in the cells, particularly the Bax and *Ku70* protein or mRNA level. In sensitizing cells to cancer therapy, the reduction is through *antisense* mRNA methods. The reduction is through inhibiting *Ku70* gene transcription, through the use of reversed full-length *Ku70* RNA, or through the use of a plasmid or viral vector encoding *antisense* *Ku70* RNA. The cells are selected from glioma cells, colon cancer cells, prostatic cancer cells, fibrosarcoma cells and cervical cancer cells. In *treating* cell death-related diseases, the increase is via the introduction and expression of heterologous DNA sequence encoding *Ku70* within the cells. These cells are selected from platelets, white blood cells and stem cells. The cells are part of an organ or are within a human patient. ACTIVITY - Cytostatic; Vasotropic. No biological data given. MECHANISM OF ACTION - Gene therapy. USE - The method is useful in modulating or examining *Ku70* levels in cells, which may be used to *treat* cell death-related diseases such as cancer and ischemia-induced cell death in nervous and cardiovascular systems, and as a diagnostic tool. (35 pages)

DESCRIPTORS: plasmid, virus vector-mediated *antisense* RNA transfer, expression in glioma, colon, prostate, cervix cancer, fibrosarcoma, platelet, white blood cell, stem cell, Bax, *Ku70* mRNA, protein expression profiling, appl. cancer, ischemia, nervous system disorder, cardiovascular disorder therapy, gene therapy, diagnosis tumor cytostatic vasotropic (22, 25)

3/3,K/9 (Item 1 from file: 399)
DIALOG(R)File 399:CA SEARCH(R)

138265632 CA: 138(18)265632g PATENT
Method of modulating or examining Ku70 and Bax levels in cells for predicting response to cancer therapy, sensitizing cells to cancer therapy and treating cell death-related diseases
INVENTOR(AUTHOR): Matsuyama, Shigemi; Sun, Wseiyong
LOCATION: USA
ASSIGNEE: Blood Center Research Foundation
PATENT: PCT International ; WO 200327237 A2 DATE: 20030403
APPLICATION: WO 2002US29737 (20020919) *US PV324292 (20010924) *US PV364287 (20020314) *US PV378585 (20020508)
PAGES: 69 pp. CODEN: PIXXD2 LANGUAGE: English
PATENT CLASSIFICATIONS:
CLASS: C12N-000/A
DESIGNATED COUNTRIES: AE; AG; AL; AM; AT; AU; AZ; BA; BB; BG; BR; BY; BZ; CA; CH; CN; CO; CR; CU; CZ; DE; DK; DM; DZ; EC; EE; ES; FI; GB; GD; GE; GH; GM; HR; HU; ID; IL; IN; IS; JP; KE; KG; KP; KR; KZ; LC; LK; LR; LS; LT; LU;

LV; MA; MD; MG; MK; MN; MW; MX; MZ; NO; NZ; OM; PH; PL; PT; RO; RU; SD; SE; SG; SI; SK; SL; TJ; TM; TN; TR; TT; TZ; UA; UG; UZ; VC; VN; YU; ZA; ZM; ZW; AM; AZ; BY; KG; KZ; MD; RU; TJ; TM DESIGNATED REGIONAL: GH; GM; KE; LS; MW ; MZ; SD; SL; SZ; TZ; UG; ZM; ZW; AT; BE; BG; CH; CY; CZ; DE; DK; EE; ES; FI; FR; GB; GR; IE; IT; LU; MC; NL; PT; SE; SK; TR; BF; BJ; CF; CG; CI; CM; GA; GN; GQ; GW; ML; MR; NE; SN; TD; TG

3/3,K/10 (Item 1 from file: 149)

02631364 SUPPLIER NUMBER: 135181438 (USE FORMAT 7 OR 9 FOR FULL TEXT)

3,4-dideoxyglucosone-3-ene induces apoptosis in renal tubular epithelial cells.

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Diabetes, 54, 8, 2424(6)
August,
2005
PUBLICATION FORMAT: Magazine/Journal ISSN: 0012-1797 LANGUAGE: English
RECORD TYPE: Fulltext; Abstract TARGET AUDIENCE: Professional
WORD COUNT: 4265 LINE COUNT: 00372

... The peptide was designed from Ku-70, a protein that suppresses the mitochondrial translocation of Bax, and it inhibits Bax-mediated apoptosis (25). Cells were *treated* with 50 (micro)mol/l 3,4-DGE for 72 h in the presence or absence of Bax inhibitor peptide P5 (200 (micro)mol/l...

...5'-TCGTCCGGCCACCGCTCACT-3', which has little complementarity with Bax mRNA but the same composition as the antisense oligodeoxynucleotide (Metabion, Martinsried, Germany) (23,27). Cells were *treated* with 50 (micro)mol/l 3,4-DGE for 72 h in the presence or absence of Bax antisense oligodeoxynucleotide (20 (micro)g/ml) or...

...DGE in tubular epithelium. We used two independent approaches to inhibit or antagonize Bax. First, the level of expression of Bax was decreased by Bax *antisense* oligonucleotides (23). Western blot confirmed that *antisense* oligonucleotides decrease Bax protein levels (not shown) (23). Decreased Bax expression protected from both apoptosis and cell death (Fig. 5). Then, Bax function was antagonized...

...Bax antagonistic peptide (25). This intervention also resulted in decreased apoptosis at 72 h (3,4-DGE: 23.7 (+ or -) 4.4%; 3,4-DGE/*Ku70* peptide: 9.7 (+ or -) 2.5%, P < 0.05 vs. 3,4-DGE; 3,4-DGE/control peptide: 20.7 (+ or -) 2.9%) and cell...

...1.08 *

Data are the mean (+ or -) SD of four independent experiments (%

apoptosis). Hypodiploid cells were quantified by flow cytometry. In all experiments cells were *treated*
with 50 (micro)mol/l 3,4-DGE for 72 h.
* P < 0.05 vs. 3,4-DGE alone; ((dagger)) P < 0.08 vs. 3...

...DESCRIPTORS: Care and *treatment*;